Cough and dyspnea - cardiogenic or non-cardiogenic?

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Cough and dyspnea, is it of cardiogenic or respiratory origin

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The following explanations focus on dyspnea but the same logic can be applied to cough. To differentiate between respiratory and cardiogenic causes, a pragmatic approach based on simple facts and logical thought processes supported by additional diagnostic tests is in most cases leading to a plausible diagnosis, which then allows the appropriate treatment. The correct conclusion is easier, when only one disease is present. More difficult are the cases, where evidence of primary cardiac and primary respiratory diseases is present, and where the difficulty consists in identifying the more important disease and likely cause of the clinical signs. This is particularly true in small breed dogs where mitral valve endocardiosis and airway collapse are commonly present in the same individual. The pressure on the clinician is higher in an emergency situation, where quick and correct treatment may make the difference between life and death. In this instance, searched is basically the answer to the question whether aggressive diuresis is indicated or not. And the very simple truth is, to diagnose cardiogenic dyspnea, we need a cardiac disease with a disease name, typical clinical abnormalities of that disease and a logical pathophysiology how this disease caused dyspnea. As a rule, the first step in the approach to a dyspneic animal is the localisation of the problem.

Simple facts

Important localisations and causes of dyspnea are:

- Upper airway obstruction, e.g. laryngeal paralysis or tracheal collapse
- Pleural disease, e.g. effusion, pneumothorax
- Pressure on the diaphragm due to intraabdominal disease, e.g. gastric distention or ascites
- Metabolic tachypnoe (may be difficult to differentiate from dyspnea), e.g. severe uremia, acidosis, hyperadrenocorticism.
- Lower airway disease (mainly in cats), bronchiole spasms
- Disease of pulmonary parenchyma, e.g. (aspiration-) pneumonia, cardiogenic and non-cardiogenic edema
- Pulmonary arteries, thrombosis

Step-by-step approach to the patient with severe dyspnea

1. no additional stress that would worsen the dyspnea. If manipulation not possible, place in quite location, give oxygen and only observe; at the same time clarify additional anamnestic questions. Criteria to observe are:
    a. respiratory rate (cave: tachypnea versus pure dead space ventilation due to panting)
    b. inspiratory dyspnea with loud stridor = suspicion upper airways
    c. predominantly very superficial fast respiration = suspicion pleural
    d. history of coughing, gagging → consider laryngeal/pharyngeal as well as lower airways.
    e. Known *relevant* heart disease = e.g. *advanced* mitral endocardiosis
    f. Duration of the disease process / respiratory signs with progression? and additional signs like lethargy, PU/PD, elevated rectal temperature, hypothermia, seizures; this for differential diagnostic thoughts like *hyperadrenocorticism* (produces severe panting, which may be misinterpreted as dyspnea, exercise intolerance, distended abdomen, all potential signs of heart failure);
    pneumonia, leptospirosis (non-cardiogenic edema) and other diseases with non-cardiogenic edema.
2. clinical findings, criteria:
    a. mucous membran color, cyanosis, too pale, too red
    b. congested jugular veins = e.g. right sided cardiac congestion, mediastinal mass
    c. heart rate, loudness of cardiac sounds, heart murmurs, arrhythmia, and pulse rate, pulse quality, pulse deficit
    d. lung auscultation, respiratory sounds muffled, where muffled, too loud, where too loud, additional sounds like crackles; careful about respiratory noises transferred from upper airways
    e. ascites, palpatory finding in abdomen

After these examinations and considerations, it should be possible to postulate true dyspnea to be present, and to raise a suspicion about its origin:

- upper airways

  Typical would be: already longer lasting respiratory problems, particularly during exercise and in hot environment, changes of the voice and the barking, cough, exercise intolerance, stridor based on history and/or during clinical examination. If

stridor and dyspnea are not present at rest, shortly exercising a dog usually allows its detection (careful, during exercise cyanosis may develop very quickly). In an emergency situation, intubation would be the life saving approach. Quite commonly, dogs with laryngeal paralysis are diagnosed as heart failure; sometimes, affected dogs, indeed, develop pulmonary edema, however, this is non-cardiogenic pulmonary edema due to low alveolar pressure.

- pleural

Typical for pleural space (pneumothorax, effusion) disease are muffled respiratory sounds in the presence of dyspnea, muffled possibly only ventrally and together with muffled cardiac sounds in pleural effusion. Pleurocentesis is the emergency treatment of choice, as a rule in the 9. intercostal space at mid-thoracic height.

- abdominal

Typical would be distended abdomen with either gas (percussion) or fluid (pear shaped belly, balottement). If fluid is suspected an abdominal tap allows differentiation between blood, infectious exsudate, chylus, modified transsudate, transsudate. A modified transsudate (protein > 25 g/l, cell count < 5000 /ul, no inflammatory or neoplastic cells) usually points to an intrathoracic and away from an intraabdominal problem.

- metabolic

Some of these cases may not be immediately differentiated from a cardiogenic dyspnea, however, if based on history and physical exams no plausible cardiac disease like mitral endocardiosis, dilated cardiomyopathy, pericardial effusion or primary arrhythmia is present, the abnormal respiration most likely is not of cardiogenic origin.

- in lung parenchyma

In this case auscultatory findings, including crackles, are expected, these, however, do not allow any conclusion about the underlying lung pathology. If signalement and history and clinical findings clearly point to decompensated heart disease, e.g. mitral valve endocardiosis, the indication for high dose furosemide is given. However, without a loud, band shaped, systolic murmur on the left thoracic side more to the apex in a small breed dog, this diagnosis is not plausible. If the parenchyma is, indeed, the problem, such a dog may rather have pulmonary fibrosis, pneumonia, pulmonary thromboembolism or a non-cardiogenic pulmonary edema.

- in pulmonary arteries

  For pulmonary thrombosis to occur it takes as eliciting cause either a primary pulmonary vascular pathology like Angiostrongylus vasorum, or a systemic prothrombotic disease like hyperadrenocorticism or nephrotic syndrome.

3. If the severity of the dyspnea does not allow to obtain thoracic radiographs, an emergency treatment must be chosen based on the above considerations. This includes
   a. oxygen,
   b. a stress free environment
   c. mild sedation, if severe excitation increases oxygen consumption aggravating the condition,
   d. pleurocentesis, if pleural disease is suspected,
   e. furosemide, if a cardiogenic cause is plausible.
4. If the dyspnea allows, inspiratory radiographs in 2 planes (LL and DV) are obtained before starting medical therapy, if the pathogenesis is not clear. Criteria to evaluate are:
   a. Diaphragmatic position, abnormalities in the (cranial) abdomen (hepatic size, ascites; if abnormalities seen, also perform abdominal rads)
   b. Presence of pleural effusion
   c. Pulmonary opacities, localisation predominantly ventral versus caudodorsal versus peripheral versus diffuse; kind of opacities, alveolar versus intersititial versus bronchial versus vascular; typical localisations are
      - (aspiration-)pneumonia ventral
      - cardiogenic edema perihilar
      - non-cardiogenic edema caudodorsal
      - Angiostrongylus vasorum peripheral
   d. Cardiac size too small versus normal versus to obig; too big more right-sided versus left-sided; to obig without obvious abnormalities in the left atrium.
      - Lung pathology combined with right-sided cardiomegaly = lung probably primary abnormality with secondary cor pulmonale
      - Lung pathology combined with left ventricular and atrial enlargement = cardiac failure is plausible cause of lung pathology.
   e. Pulmonary vessels too small, too large; arteries relative too large, veins relative too large.

- arteries and veins (too) small = left-sided cardiac failure essentially ruled-out as cause for dyspnea.
- Arteries relative too large = probably primary lung problem
- Veins too large = left sided heart failure plausible.

5. Decision, whether cardiogenic versus non-cardiogenic dyspnea, and based on this decision and underlying disease emergency treatment.

Cardiogenic dyspnea in the form of pulmonary edema needing aggressive furosemide therapy is probably present if the following are true:

- „typical“ clinical signs of a plausible cardiac disease,
    - Loud heart murmur of mitral insufficiency, murmur typically known for a long time and getting progressively stronger; cough, typical for advanced mitral endocardiosis.
    - Rather low intensity murmur in larger breed dogs combined with tachyarrhythmia (atrial fibrillation, ventricular premature contractions), rather weak pulse, suggestive of dilated cardiomyopathy.
    - Continuous murmur high on the thorax, typical for a PDA.
- Combined with typical radiographic changes, i.e. left-sided cardiomegaly with large left atrium, dorsal displacement of trachea and stem bronchi, combined with perihilar or dorso-caudal interstitial lung pattern.

It shall be mentioned that most of the respiratory emergencies are not caused by mitral valve endocardiosis or dilated cardiomyopathy. Thus, in most respiratory emergency cases furosemide is not indicated.

References

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